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Indoor Environmental Determinants of Lung Function in Children

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16. ABSTRACT

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Indoor Environmental Determinants of Lung Function in Children[1,2]

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SUMMARY Using pulmonary function and family respiratory questionnaire data for 16,689 white children 6 to 13 yr of age from 7 geographic areas, the investigators examined the effect of several environmental and other factors on performance in a standard test of breathing. As expected, $FEV_{0.75}$ was correlated most strongly with age, height, and sex. A dose-response relationship was observed with maternal smoking habits and explained 0.1% of the variance. No effect caused by the father's smoking habits was observed. A decrease ($p = 0.0524$) in FEV among older girls was associated with the presence of a gas cooking stove in the home. Although the statistical significance of the decreases was largely attributable to the size of the sample, the decreases in FEV, even though small, were thought to be biologically significant.

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Introduction

Lung function in children has been related to both genetic and environmental factors. Genetic factors exert the greatest influence through general stature, as measured by height and by age. Environmental exposures include both those ambient in origin and those attributable to indoor sources such as smokers and gas stoves. This report discusses the association of indoor environmental pollutants and children's lung function.

It is well known that familial factors are important predictors of respiratory disease (1-6). Investigators have observed an excess prevalence of chronic bronchitis in relatives of persons with bronchitis when compared with relatives of persons without bronchitis (1-3). More recent studies have noted the household clustering of bronchitis and described its genetic component (4-6).

Genes, however, are only one thing that a family has in common; a large amount of time spent breathing a common atmosphere is another. Estimates vary, but children living in a temperate zone are thought to spend 60 to 80% of their time indoors on an average school day (7). Thus the presence of known sources of pollution (e.g., smokers and gas stoves) inside the home where reduced ventilation can lead to pollutant build-up may expose children to potentially harmful environments.

The impact of smoking on indoor atmospheres has been demonstrated numerous times (7-9). For example, under average indoor conditions, concentrations of particles in the respirable range (<3.5 microns in diameter) can exceed the National Ambient Air Quality Standard for total suspended particulates (all particles <30 microns in diameter) (9).

Several studies have reported that exposure of infants to side-stream smoke from parental smoking increases the risk of an attack of pneumonia or bronchitis (10-12), but this association was not observed in children 1 to 5 yr of age. Speizer and co-workers (13) found an association between parental smoking and a history of respiratory disease in children younger than 2 yr of age. Colley (14) reported that the prevalence of cough in children 6 to 14 yr of age was associated with their parents' smoking habits and that the prevalence was highest when both parents smoked and lowest when neither parent smoked.

The literature regarding effects of passive smoking on lung function is limited. Leeder and associates (15) observed no influence of parental smoking habits on peak expiratory flow rates of children 0 to 5 yr of age, even though parental smoking was associated with the incidence of bronchitis in the first year of life. In the same cohort, bronchitis in the first year of life was associated with decreased expiratory flow rates. By contrast, Tager and colleagues (16) recently reported that children 5 to 19 yr of age showed patterns of decline in forced expiratory flow during the middle half of the FVC ($FEF_{25-75\%}$) that were indicative of a dose-response relationship with parental smoking. However, no association of parental smoking with respiratory illness was observed in these children.

In addition to passive smoking, the health significance of the gas-fueled stove is also becoming apparent. Numerous studies have demonstrated that gas stoves contribute significantly to indoor nitrogen dioxide concentrations (17-21). Although experiments involving animals have suggested an association between nitrogen dioxide and respiratory infections (22-24), the epidemiologic evidence does not disclose a consistent relationship between NO_2 exposure from gas stoves and health effects. Melia and co-workers (25) conducted a longitudinal study examining the relationship of cooking fuel and respiratory disease in children. They concluded that girls from homes using gas for cooking reported more respiratory symptoms or diseases than were reported by girls from homes where electricity was used. Speizer and co-workers (13) found children 6 to 10 yr of age in homes equipped with a gas stove to have significantly lower FEV_1 and FVC values and to have a greater history of respiratory illness before 2 yr of age, when compared with children of the same age in homes with electric stoves. By contrast, Keller and colleagues (26) compared gas and electric cohorts of school-age children. The data revealed no differences in reported incidence of respiratory illnesses, the frequency of symptoms, or the fre-

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quency of positive respiratory findings on physical examination.

Methods

The data for this study were collected between 1970 and 1973 as part of the Environmental Protection Agency's Community Health Environmental Surveillance System (27). We were careful to select for this report only data that were comparable with those in the existing epidemiologic literature. Potential methodological problems in the data are discussed where needed.

Community selection. Communities were chosen from 7 metropolitan areas in different geographical regions of the continental United States (table 1). Ambient air pollution measurements were obtained from 1971 to 1973, which provided data for total suspended particulates, respirable suspended particulates, and the water-soluble sulfate fraction of the total suspended particulates. Monitoring of indoor atmosphere was not a part of this program, but relative exposures to NO_2, total suspended particulates, and respirable suspended particulates can be inferred from the presence or absence of gas stoves (19–21, 26) or smokers (7, 28, 29) in the house.

Questionnaire administration. Within each community, children in the elementary schools (5 to 13 yr of age) were enrolled (table 1). A family health questionnaire, based on the British Medical Research Council's Chronic Bronchitis questionnaire, was distributed via the youngest child from each family. From this questionnaire, current parental smoking habits, family socioeconomic status, and other information were obtained (27). Where possible the mother or female guardian was asked to complete the form and return it to the school for collection. In all areas except Utah, questions regarding the nature of cooking and heating in the home were asked. For the children recruited, data on personal smoking habits and respiratory illness histories (other than asthma) were not collected.

Questionnaire distribution covered a 2-yr period with pulmonary function testing taking place during the 1972–1973 school year. The questionnaire data was at times collected in advance of the testing (for exact distribution dates, see table 1). In New Jersey, for example, questionnaires were distributed between 15 and 24 months prior to pulmonary function testing. Parental cooperation, averaged over all areas, was approximately 85%.

Pulmonary Function Testing. Pulmonary function testing was performed in the schools with a 12-L bellows-type spirometer manufactured by Cardiopulmonary Instruments, Inc. (Houston, Texas). In this instrument, expired air displaces a cylinder whose air seal is a pliable rolling diaphragm. The mechanical displacement of the piston over 0.75 s was transduced to a voltage that was displayed as $FEV_{0.75}$ (forced expiratory volume in 0.75 seconds) on a nixie-tube digital readout.

The study design allowed for only $FEV_{0.75}$ measurements to be obtained. Using the digital display of the $FEV_{0.75}$ precluded the

TABLE 1

SAMPLE SIZES, DATES, AND EXCLUSIONS BY COMMUNITY

Community	Original Number Surveyed	Number Nonwhite	Number Asthmatics	Number with <2 Tests*	Number with No Background Information	Number with Complete Information on Both Parents	Dates of Background Questionnaire
Charlotte NC							
Sector 2	2,605	1,066	77	504	332	556	10/72–12/72
Sector 3	2,524	954	82	569	402	441	10/72–12/72
Sector 4	2,301	754	55	522	92	771	10/72–12/72
Birmingham AL							
Inglenook	1,270	189	72	369	121	458	10/72–12/72
Norwood	2,062	1,879	13	87	30	44	10/72–12/72
Zion City	1,008	221	56	262	148	279	10/72–12/72
Center Point	1,994	225	75	446	60	1,118	10/72–12/72
Homewood	1,412	155	69	341	33	695	10/72–12/72
Hueytown	1,722	285	110	403	61	796	10/72–12/72
Riverhead NY	1,402	496	35	322	187	309	5/72
Queens NY	1,272	103	41	531	157	411	5/72
Bronx NY	1,324	309	34	410	204	318	5/72
Sheepshead Bay NY	1,003	124	32	305	125	361	5/72
Ridgewood NJ	1,434	58	41	473	282	554	6/71
Fair Lawn NJ	1,270	27	40	419	210	549	6/71
Matawan NJ	1,385	35	51	439	259	582	6/71
Elizabeth NJ	1,372	176	30	450	181	464	6/71
Ogden UT	1,259	120	42	389	440	239	12/70
Salt Lake City UT	1,971	340	57	642	480	377	12/70
Kearns UT	2,007	167	73	734	505	485	12/70
Magna UT	2,052	244	69	576	476	619	12/70
Chattanooga TN							
Red Bank	1,878	10	65	594	607	551	1/72–3/72
Brainerd	2,146	686	65	469	425	432	1/72–3/72
Harrison	2,125	206	61	622	618	571	1/72–3/72
Vista CA	2,479	573	138	512	434	736	12/71–3/72
Santa Monica CA	2,295	587	138	498	412	472	12/71–3/72
Anaheim CA	2,555	271	180	663	591	726	12/71–3/72
Glendora CA	2,245	125	160	540	496	865	12/71–3/72
Thousand Oaks CA	1,902	106	131	535	374	712	4/72–5/72
Garden Grove CA	2,180	284	136	654	409	593	4/72–5/72
Covina CA	2,414	378	151	686	501	605	4/72–5/72
Total	56,868	11,153	2,379	14,964	9,652	16,689	

* Includes those absent or those present with cough, cold, or sore throat, twice or more.

correction of each reading for submaximal effort at the beginning of a test. The values reported here are actually for FEV of less than 0.75 s. The average amount of time less than 0.75 s and the reduction of air volume associated with it were unobtainable from our data.

The cardiopulmonary instrument was calibrated before testing each day against both a Collins water-filled spirometer and a 1.5-L syringe. Several times each testing day the linearity of the digital readout was checked. To counter potential technician differences, an average of all blows from more than one period was taken. All raw $FEV_{0.75}$ readings were converted to body temperature and pressure-saturated conditions (BTPS). Different technicians and machines were used in each metropolitan area.

Pulmonary function testing was conducted seasonally during the school year for a different number of years in each area (table 1). To control for possible annual differences in testing procedures, only tests from the 1972–1973 school year have been used in the following analyses. Three rounds of tests were conducted that year: fall 1972, winter and spring 1973. Just before testing, the children received an introduction and test demonstration that emphasized maximal inspiration first, then expiration, as forceful and complete as possible, into the spirometer. Each child was tested until 3 acceptable FEV measurements were obtained. The maximum of each child's 3 readings was used in the data analysis. At the time of testing, each child reported the presence or absence of a cough, cold, or sore throat.

Each technician was responsible for selecting an acceptable test. To check technician selection accuracy, an average was taken of each technician's 3 (acceptable) recorded FEV values. All means obtained for each day of testing were averaged for every technician. The technician supervisor was to correct any technician who consistently produced a mean score that differed from the mean scores of other technicians.

Sample selection. The original file contained 56,864 children who participated at any time in the 1972–1973 school year (table 1). To avoid possible confounding by race (28), analyses were restricted to the 45,711 white children. Any child reported to have ever had asthma was excluded. Only children free of respiratory symptoms in at least 2 test periods were included. This left 28,366 children eligible for analysis.

In order to investigate the association of parent-reported socioeconomic and exposure information with FEV values, it was necessary first to link the parents' questionnaire information to the data from the children's pulmonary function tests. This proved to be difficult. Although there were problems with record keeping, most of the difficulty was attributable to the interval between questionnaire distribution and pulmonary function testing. As a result of this interval, linkage was not possible for those eligible who had moved out of study communities (after questionnaire distribution but before testing) and those eligible who had advanced to an age that disqualified them from participating. The average linkage achieved for all areas was approximately 65%, which reduced the sample to 18,716. The sample was further reduced by restricting the analysis to students for whom smoking information on both parents was available. The final sample total, then, included 12,748 families with 16,689 white, symptom-free children tested in at least 2 of the 3 test periods and for whom questionnaire information was available on both parents.

Analytical methods. FEV was analyzed using a linear model. The model included terms for height, age, sex, community of residence, educational attainment of head of household, season of missing FEV, and smoking status of parents. In particular, for child "i" in family "j" the model was

$$FEV_{ij} = B_1\,height_i + B_2\,age_i + B_3\,sex_i + B_4\,comm_j + B_5\,educ_j + B_6\,season_i + B_7\,smoke_j + \varepsilon_j + \delta_i$$

where $comm_j$ represented a term (dummy variable) for the residence of the family; $smoke_j$, a term for the smoking status of each parent; $educ_j$, a term for educational attainment of the head of household; $season_i$, a term for possible season of testing missed; ε_j represented the variation of FEV from family to family, and δ_i represented the variation of FEV within a family. The dummies were constrained so that the design matrix was nonsingular.

The above model was equivalent to stating that the FEV values could be analyzed using analysis of covariance. Age and height were the covariates, and sex, community, education, season, and parents' smoking habits were the main effects. Because the design was an unbalanced one, the analysis had to be done using a linear model program. The model as stated required the inversion of a 75 by 75 matrix. Interaction terms were not included because the main effects were relatively small, and the interaction terms would have greatly increased the computational difficulties.

The error sum of squares were split into 2 portions: a between-family variation and a within-family variation. The tests of significance were made using the between-family variation. Because the design was not balanced, these tests were not exact. However, since the majority of families had only one child tested, the tests were approximately correct.

Additional analyses were restricted to those families who answered questions on the presence of a gas stove in the home. Both the effect of maternal smoking and the presence of a gas stove were estimated separately for boys and girls younger than 9 and those between 9 and 13 yr of age.

Results

The results of the initial analysis of the $FEV_{0.75}$ data are shown in table 2. The factors included in the model explain 79% of the total variation, with age and height alone accounting for 73.1% of the variation. The relationship of $FEV_{0.75}$ with both age and height was almost linear over the range of ages and heights observed. The relationships were almost identical to those found by Hamill (29) in his study of vital capacities in children 6 to 11 yr of age. A fairly constant sex difference was observed, with boys showing average values 91 ml larger for the same age

TABLE 2

ANALYSIS OF COVARIANCE FOR CHILDREN WITH BACKGROUND INFORMATION ON BOTH PARENTS

Factor	Degrees of Freedom	Partial Sums of Squares	Mean Square	Approximate F*	Approximate p-value
Age-height-sex adjustments	5	2,019.16	403.8323	19,843.96	< 0.0001
Community	30	22.27	0.9422	25.53	< 0.0001
Education	2	0.05	0.0265	0.72	0.4863
Season missing	3	0.15	0.0509	1.38	0.2500
Parents smoking	35	2.68	0.0765	2.07	< 0.0001
Interaction	25	1.35	0.0542	1.49	0.0520
Fathers	5	.01	0.0014	0.04	0.9991
Mothers	5	1.33	0.2655	7.20	< 0.0001
Packs (mother)	1	1.21	1.2131	32.88	< 0.0001
Other	4	0.12	0.0288	0.78	0.5058
Error (total)	16,613	567.17	0.0341		
Between family	12,748	470.44	0.0369	1.48	< 0.0001
Within family	3,865	97.73	0.0250		

Multiple R^2 = 0.78978

Prediction equations:

Boys $FEV_{0.75}$ = 2.274 + 0.0231 age (yr) + 0.0267 height (cm.)

Girls $FEV_{0.75}$ = 2.239 + 0.0246 age (yr) + 0.0261 height (cm.)

and height. The prediction equations for boys and girls are given in table 2.

Pulmonary function test results have been shown to vary seasonally (30, 31). Therefore, since each child was required to be in the analysis only 2 of the 3 seasons, additional terms were added to adjust for missing seasons. This result was not statistically significant, although children who were tested all 3 seasons tended to have higher FEV values than those who missed a season.

Using educational attainment of head of household and density of people per room alternatively as indexes of socioeconomic status did not affect FEV values. Socioeconomic variation, as measured by these factors, did not appear important.

The variation between families was about 50% greater than the variation within families. This showed clear evidence of familial aggregation. The estimated intraclass correlation was 0.26 ($p < 0.0001$).

Of the remaining factors, differences between the 31 communities accounted for the greatest amount of variation. Ambient air pollutant variables were included in an attempt to explain the sums of squares labeled community variation. No pollutant explained more than 8% of the community variation. Community mean values adjusted for age and height are shown in table 3. Possible explanations for this variation are considered subsequently.

TABLE 3

MEAN $FEV_{0.75}$ MEASUREMENTS BY COMMUNITY ADJUSTED FOR AGE AND HEIGHT

Community	Boys 6-9	Boys 9-13	Girls 6-9	Girls 9-13
Charlotte NC				
Sector 2	1.320	1.862	1.252	1.792
Sector 3	1.335	1.874	1.232	1.795
Sector 4	1.368	1.883	1.278	1.826
Birmingham AL				
Inglenook	1.275	1.745	1.147	1.633
Norwood	1.367	1.715	1.108	1.682
Zion City	1.261	1.799	1.158	1.684
Center Point	1.289	1.809	1.190	1.712
Homewood	1.303	1.821	1.217	1.716
Hueytown	1.228	1.781	1.129	1.669
Riverhead NY	1.304	1.787	1.168	1.664
Queens NY	1.269	1.744	1.160	1.580
Bronx NY	1.294	1.793	1.194	1.693
Sheepshead Bay NY	1.254	1.768	1.171	1.624
Ridgewood NJ	1.295	1.750	1.191	1.708
Fair Lawn NJ	1.270	1.733	1.140	1.641
Matawan NJ	1.275	1.782	1.162	1.705
Elizabeth NJ	1.229	1.777	1.166	1.712
Ogden UT	1.378	1.921	1.257	1.860
Salt Lake City UT	1.365	1.889	1.252	1.824
Kearns UT	1.319	1.885	1.226	1.752
Magna UT	1.387	1.881	1.224	1.766
Chattanooga TN				
Red Bank	1.291	1.838	1.190	1.740
Brainerd	1.300	1.828	1.170	1.690
Harrison	1.277	1.781	1.168	1.683
Vista CA	1.317	1.858	1.220	1.734
Santa Monica CA	1.356	1.847	1.256	1.802
Anaheim CA	1.325	1.844	1.232	1.746
Glendora CA	1.341	1.865	1.224	1.755
Thousand Oaks CA	1.350	1.838	1.224	1.769
Garden Grove CA	1.317	1.826	1.236	1.756
Covina CA	1.326	1.855	1.224	1.747

The only other highly significant factor in the analysis was parental smoking, which accounted for 0.1% of the total variation. Each parent was placed in 1 of 6 categories on the basis of the amount he or she smoked. An analysis of ex-smoking parents showed that their offspring's average FEV was virtually identical with the nonsmokers, so the 2 groups were pooled. The 6 remaining categories were based on the amount currently smoked: 0, <0.5 pack, 0.5 to <1 pack, about 1 pack, >1 to 1.5 packs, and 2 packs or more. The 6 categories of smoking for each parent gave 36 possible combinations. The sums of squares for these were split into 3 categories: the amount the mother smoked, the amount the father smoked, and the interaction of the 2. Neither the interaction nor the amount the father smoked were significant at 0.05. The mother's amount, however, was highly significant.

The mothers' amount was split into 2 components: a linear pack function, and a nonlinear component. The linear pack function explained nearly all of the variation (91%), with an insignificant amount remaining in the nonlinear portion. Thus, parents' smoking could best be represented by the simple function of the amount smoked by the mother. This function was used in further analyses.

The effect of maternal smoking appeared in both sexes and age groups (table 4). The effect on older boys was greater than it was on the children in the other 3 subgroups. The presence of a gas stove in the home had a less dramatic effect (table 4), with the older girls showing a decrease that approached statistical significance ($p = 0.0524$). It is interesting to point out (table 4) that the effect on boys in homes with gas stoves was slightly greater, although not significantly so. The means for the presence of a gas stove were adjusted for community differences so that the comparisons would be within communities, as was done by Speizer and coworkers (13).

Discussion

Parental smoking. These data indicated a negative association between the amount smoked by the mother and the $FEV_{0.75}$ value of the child living in her home. Similar evidence of a crude dose-response relationship between $FEF_{25-75\%}$ and the number of smokers in a house has been reported by Tager and colleagues (16). Our data revealed only a slight negative and insignificant influence of the amount smoked by the father on the results of his child's pulmonary function test, after considering the amount smoked by the mother. This difference in results might be explained by the method used to gather parental smoking information: the Tager study obtained smoking information by direct interview with each parent, whereas in these data, smoking information was obtained by

TABLE 4

MEAN $FEV_{0.75}$ MEASUREMENTS ADJUSTED FOR AGE AND HEIGHT SUMMARIZED BY COVARIATES

	Boys 6-9	Boys 9-13	Girls 6-9	Girls 9-13
Mother's smoking				
Never or ex	1.315	1.833	1.211	1.734
	(1,933)*	(2,949)	(1,777)	(2,408)
Less than 1 pack	1.301	1.825	1.195	1.729
	(650)	(890)	(597)	(772)
1 or more packs	1.294	1.806	1.195	1.722
	(1,016)	(1,470)	(917)	(1,310)
Gas stove use				
Yes	1.309	1.829	1.203	1.719
	(1,173)	(1,454)	(1,087)	(1,208)
No	1.304	1.825	1.208	1.738
	(1,221)	(2,098)	(1,178)	(1,792)
Education of head of household				
Less than high school	1.296	1.830	1.199	1.739
	(623)	(955)	(531)	(821)
High school	1.306	1.824	1.203	1.728
	(2,121)	(3,040)	(1,926)	(2,593)
More than high school	1.317	1.820	1.209	1.727
	(855)	(1,314)	(834)	(1,076)

* Sample sizes given in parentheses.

a self-administered questionnaire and was reported for both parents by only one parent (usually the mother). It may well be that this method of data collection left us with smoking data that was more accurate for the mothers (self-reported) than for the fathers (reported by another person). Although conjectural, this reasonably could account for the low explanatory power of our father-smoking factor: alternatively, the greater significance of a mother's smoking on the results of her offspring's test may be due to the greater time spent with the child.

Other studies have not found an association between parental smoking and test performance by children. Leeder and associates (15) found no effect on peak expiratory flow rates. This was somewhat inconsistent with our results since our flow rates were strongly correlated with $FEV_{0.75}$ (32, 33), but the investigators tended to attribute this result to small sample size. In Shilling and co-workers (34), although the results suggested a relationship between the amount the mothers smoked and maximal expiratory flow at 50% of FVC among their daughters (if they had never smoked), the investigators concluded parental smoking had no effect on the children's lung function. Finally, Speizer and co-workers (13), working with 8,120 white children between 6 and 10 yr of age, did not find an association between FEV_1 and parental smoking. This finding was possibly due to the age of the subjects. In young children, the FEV_1 may approximate the FVC and be better correlated with lung volume than the $FEV_{0.75}$ used in the present study. We know of no data that support this supposition. The $FEV_{0.75}$ may be more sensitive to variation in airway obstruction, which would suggest that sidestream smoke may have a more obstructive than restrictive effect on the lungs. For a discussion of possible mechanisms, see Tager and colleagues (16).

The Tager results are interesting with regard to the findings of Colley reporting both alone (11, 14) and with Leeder and associates (12) the relationships between the number of smoking parents in a household and the number of respiratory diseases among their children. Other reports, however, are not consistent with the Colley findings. Schilling and co-workers (34) and Lebowitz and colleagues (35) found little or no relationship between smoking parents and the number of reported respiratory symptoms or diseases among their children. They suggested that familial characteristics, such as parent symptoms and family history of respiratory ailments, rather than parental smoking, more readily influence the number of reported respiratory symptoms among children. It is also important to note that within the Tager and colleagues study (16), the investigators did not report a dose-response effect between the number of smoking parents and the number of illnesses in their children.

Gas stoves. Speizer and co-workers (13) using FEV_1 did report a negative association between exposure to a gas cooking stove and the results of a child's pulmonary function test. Our data were less consistent but did show an association that approaches statistical significance of gas stove use and reduced $FEV_{0.75}$ among the sex/age group in which exposures can reasonably be expected to have been highest, e.g., girls 9 to 13 yr of age (table 4). This concentration of effect among females was consistent with the findings of Melia and co-workers (25) who suggested that girls would be more likely to help in the kitchens of their homes—the room with the highest NO_2 exposure in the house (19).

There are 3 possible explanations for these findings. First, older girls may be in the kitchen longer than younger girls. Alternatively, the accumulative length of NO_2 exposure may be more important than simple frequency of exposure. Third, a possible interaction may exist between smoking and NO_2 exposure in older girls. As there was no direct measurement of smoking by children, this last alternative is difficult to assess. In any case, the observed changes were small. However, as set forth by Speizer and co-workers (13), even small differences in age- and height-adjusted lung function in young children may indicate that these children will not develop full lung size in adulthood.

These findings were in contrast to Florey and associates (36) who found that the lung function of 808 school-age children, as measured by $FEV_{0.75}$, peak expiratory flow rate, and the mean flow over the mid-half of the FVC, was not related to NO_2 concentrations in their bedrooms or kitchens. It is also important to note that Melia and colleagues (37), although finding respiratory disease and symptom prevalence higher in homes using gas for cooking instead of electricity, concluded that the differences were limited to urban areas and might disappear as the children grew older. Similarly, Keller and co-workers (26) found no differences in reported illnesses, symptoms, or positive findings on physical examination when they compared gas and electric cohorts of school-age children.

Other factors. A significantly greater

amount of the sums of squares attributable to error was found between families than within families (table 2). It was possible, as has been suggested by others (14, 34), that these differences may have been a result of within-family reporting bias. This may be a problem for analyses that consider smoking information, as has been reported by Colley (14). According to Speizer and co-workers (13), however, it is unlikely that within-family reporting bias would be related to the presence of a gas or an electric stove. Our results supported the findings of Speizer and associates (4) and Tager and colleagues (5, 6), which showed that lung diseases tend to aggregate within families.

There were several other potential sources of bias in these data. As mentioned before, the amount the father smoked may be confounded by the reporting method used. It may artifactually have minimized the importance of the father's smoking behavior in our study, whereas it has been found to be significant by other researchers.

Questionnaire information regarding parental smoking and gas stoves could not have influenced technician performance with each child as this information was gathered separately from the pulmonary testing. Regular (daily) calibration of spirometers helped minimize machine biases, but examination for machine bias in some of our sites in other test years revealed that such biasing did occasionally occur. Machine bias, however, was not consistent for any machine in more than a single testing season and, thus, its effect was probably minimal. Bias caused by technician differences should have been countered by the averaging of blows from more than one period. This averaging would have allowed for both a training effect and mixing of technicians period to period. Any remaining technician or machine biases would show up as community differences since different technicians and machines were used in each metropolitan area.

Certain other information would have helped reduce bias and sharpen the distinctions between the exposed and unexposed groups. Unfortunately, children examined in this study were not asked about their personal smoking habits. Confounding could be a problem if the children of smoking parents themselves tended to smoke; such an association has been reported (38). Possible evidence of this confounding is found in the greater reduction of FEV observed among the older males in our sample, the group that in the early 1970s could be expected to have been smoking most heavily (38). A less likely explanation would be some sort of greater susceptibility of males to side-stream smoke, a susceptibility that would increase with age. Personal smoking is an unlikely explanation for the reduced FEV observed among those 6 to 9 yr of age.

Although no one has assessed the reduction of FEV among smoking children, Tager and colleagues (16) have shown that the maximal $FEF_{25-75\%}$ reduction among smoking children is greater than the $FEF_{25-75\%}$ among children whose parents smoke. Though there are implications from Tager's findings for the interpretation of our results, there are no studies to date assessing the relative impact of children's smoking habits on their FEV versus the effect of the smoking habits of their parents.

In addition to the bias introduced as a result of the linking of the questionnaire and the pulmonary function test data, misclassification of parental smoking is expected to have occurred because of the interval between questionnaire distribution and pulmonary testing. Parents reporting themselves to be nonsmokers may have begun to smoke or vice versa, or the amount of smoking reported may have changed. Furthermore, the amount of present exposure may be less important than the duration of exposure to that amount of side-stream smoke or the total accumulated exposure of a child over his or her youth. None of these questions can be addressed with these data.

Certain points regarding our FEV values should be made. First, to facilitate the testing of a large number of children the digital display of $FEV_{0.75}$ was used. Unfortunately, the digital display of the $FEV_{0.75}$ does not allow for a correction of submaximal effort at the beginning of a test. In a large-scale epidemiologic study, the use of a visual record to evaluate test performance is problematic. The technological advancement of the spirometer since our data collection, however, has alleviated many of the difficulties. Gaensler (39), in discussing procedures for pulmonary function testing in epidemiology, made a number of detailed recommendations regarding the use of the spirogram (or other record) to evaluate test performance. Second, our recording of only the highest value in each test period favored selection of tests only minimally influenced by submaximal effort. The averaging of values from more than one test period would also allow for a training effect to help minimize this (submaximal) effect. Comparison of these values with $FEV_{0.75}$ readings from machines that allow correction should show the latter values to be somewhat higher. Third, other information, which was not available from these data, such as the variation in all FEV values for each child, the effect on the community, maternal smoking, or gas stove effect by including only children free of respiratory symptoms, the adequacy of the correction for seasonal effect, the amount of mixing of machines and technicians, and the variation found when calibrating the instruments each day, would have helped assess the reliability and validity of the spirometry data.

In summation, the effect of parental smoking on a child's breathing has been observed. The impact of a mother's smoking shows a clear dose-response relationship with a small but significant reduction in her child's ability to exhale air quickly from his or her lungs. The importance of the father's smoking habits is uncertain. The effect of gas stove exposure, which approached statistical significance, was observed in the group most likely to be at risk of highest exposure—older girls.

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